

Genetic make-up influences biased economic decision-making, study shows

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How would you respond if you were told that you had an 80% chance of surviving an operation - would you give consent? How about if you were told you had a 20% chance of dying? The answer may partly depend on your genetic make-up, according to new research from UCL (University College London) and funded by the Wellcome Trust.

Decision-making is a complex process, particularly when we are uncertain about outcomes. This makes decisions open to influence depending on whether the options are phrased positively or negatively, known as the "framing effect".

Previous research from the Wellcome Trust Centre for <u>Neuroimaging</u> at UCL suggested that the amygdala, an area of the brain known to be involved in processing emotions, becomes active during decisions influenced by the framing effect. Now, in a study published today in the *Journal of Neuroscience*, UCL researchers have shown that a person's susceptibility to the framing effect - and the response of their amygdala appears to be at least partially influenced by their genetic make-up.

"We know that people from across a variety of cultures are susceptible to biases when making decisions, and that even with training these biases are hard to overcome," says Dr Jonathan Roiser from the UCL Institute of <u>Cognitive Neuroscience</u>. "This implies that hard-wired genetic influences might play an important role in determining how susceptible different individuals are to the framing effect."



In this new study, Dr Roiser and colleagues showed that decision-making is affected by variation in the <u>serotonin transporter gene</u>, at a region known as the 5-HTTLPR, which has previously been reported to affect the response of the amygdala. The gene is involved in the recycling of serotonin, a neurotransmitter essential for communication between <u>nerve</u> <u>cells</u>. The researchers investigated two common variants of this gene, known as the "short" and "long" versions. The researchers selected thirty healthy volunteers carrying either a pair of short variants or a pair of long variants.

Participants in the study performed a task involving deciding whether or not to gamble with a sum of money. For example, they would be given £50 and be presented with two options: option A was to keep £20 for sure, while option B was to gamble, with a forty per cent chance of keeping the full £50 and a sixty per cent chance of losing everything. This version was known as the "gain frame".

At other times, the participants were presented with exactly the same decision, but framed differently - the "loss frame". The only difference was that option A was phrased in terms of losing money. In other words, after being given £50, option A was to lose £30 of their initial amount for sure, while option B was the same gamble as above.

Despite option A representing an identical decision in the gain and loss frames - which all of the volunteers realised - the researchers found that both groups of participants were more likely to gamble if the first option was phrased in terms of losing rather than keeping money. The magnitude of this difference in gambling between the two frames essentially measures each volunteer's susceptibility to the framing effect. Critically, those participants with two copies of the short variant were considerably more susceptible to the framing effect.

"This doesn't mean that people with the short variants are risk takers,"



explains Dr Roiser. "In fact, they were risk averse in the 'gain frame' whilst risk seeking in the 'loss frame', which implies inconsistency in their decision-making."

Brain images taken while participants made their decisions revealed a mechanism underlying this difference in decision-making behaviour. Participants with two copies of the short genetic variant had greater amygdala responses than their counterparts when making decisions influenced by the frame effect.

The researchers also measured the degree of interaction, or connectivity, between the amygdala and the prefrontal cortex, the brain region most implicated in human intelligence, personality and decision making. When resisting the frame effect, the participants with two copies of the long variant had stronger connectivity between the prefrontal cortex and amygdala, while those with a pair of short variants did not.

"This difference in connectivity is really interesting," says Dr Roiser. "It suggests that the volunteers carrying the long variant might regulate automatic <u>emotional</u> responses, which are driven by the amygdala, more efficiently, lessening their vulnerability to the framing effect.

"This one gene cannot tell the whole story, however, as it only explains about ten per cent of the variability in susceptibility to the framing effect. What determines the other ninety per cent of variability is unclear. It is probably a mixture of people's life experience and other genetic influences.

"An interesting question would be whether the gene might affect reallife <u>decision-making</u>. For example, traders in banks need to make quick and accurate estimations of risk and consistent decisions, no matter how the information is presented to them. So you might hypothesise that traders with the long genetic variant would make more consistent



decisions, though this needs to be tested in future research."

Source: Wellcome Trust (<u>news</u> : <u>web</u>)

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